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Symbol	Name	Synonyms	Organism
CDC25		Cell division control protein 25, CTN1, L2142.6, YLR310C	Saccharomyces cerevisiae

UniProt P04821
NCBI Gene 851019
NCBI RefSeq NP_013413
NCBI UniGene 851019
NCBI Accession AAB64528, CAA27259

Homologues of CDC25 ... new

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Extensive information has been obtained on the core section of the pathway, i.e. **Cdc25**, Ras, **adenylate cyclase**, PKA, and on components interacting directly with this core section, such as the Ira proteins, Cap/Srv2 and the two cAMP phosphodiesterases.

The SH3 domain of the S. cerevisiae **Cdc25**p binds **adenylyl cyclase** and facilitates Ras regulation of cAMP signalling.

These studies suggest that a direct interaction between **Cdc25** and **adenylyl cyclase** promotes efficient assembly of the **adenylyl cyclase** complex.

Cdc25 is essential for Ras-mediated activation of **adenylyl cyclase** in the yeast Saccharomyces cerevisiae.

It is also shown that 6-deoxyglucose can activate **adenylate cyclase** in the absence of **CDC25** gene product.

The activation of **adenylate cyclase** by **guanine nucleotides** and 6-deoxyglucose was studied in membrane preparations from S. cerevisiae mutants lacking the **CDC25** gene product.

Activation of **adenylate cyclase** in **cdc25** mutants of Saccharomyces cerevisiae.

The relative amount of membrane-bound **adenylate cyclase** was drastically reduced in **cdc25** ts membranes when subjected to the restrictive temperature, while no significant change was observed in the wild type.

Adenylate cyclase from **cdc25** ts membranes was activated by GTP and GppNHp in membranes from cells collected after **glucose** was exhausted from the medium.

These results indicate that the **CDC25** gene product is required not only for basal cAMP synthesis in yeast but also for specific activation of cAMP synthesis by the signal transmission pathway leading from **glucose** to **adenyl cyclase**.

Overexpression of the gene **CDC25** in the ras1ras2bcy1 strain

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by Robert Hoffmann

relocalizes **adenylyl cyclase** activity to the membrane fraction.

The reconstitution experiments described provide direct biochemical evidence for the role of the **CDC25** protein in regulating the RAS dependent **adenylyl cyclase** in S.cerevisiae.

In vitro reconstitution of **cdc25** regulated S. cerevisiae **adenylyl cyclase** and its kinetic properties.

This modulation requires functional elements of the cAMP-producing pathway, **adenylate cyclase**, ras proteins and the product of **CDC25** gene.

In the yeast Saccharomyces cerevisiae, the activation of **adenylate cyclase** requires the products of the RAS genes and of **CDC25**.

We propose that **CDC25** regulates **adenylate cyclase** by regulating the guanine nucleotide bound to RAS proteins.

Cells lacking **CDC25** have low levels of cyclic AMP and decreased levels of Mg2+-dependent **adenylate cyclase** activity.

The activation of **adenylate cyclase** by guanyl nucleotides in Saccharomyces cerevisiae is controlled by the **CDC25** start gene product.

In the thermosensitive **cdc25** start mutant of Saccharomyces cerevisiae, the regulation of **adenylate cyclase** by guanyl nucleotides was rapidly nullified when the enzyme was prepared from nonsynchronized cells shifted to the restrictive temperature.

top

In view of the likely involvement of the **CDC25** protein in the regulation of **adenylate cyclase** activity, a working hypothesis is proposed that accounts for the observed homologies.

The N-terminal half of **Cdc25** is essential for processing **glucose** signaling in Saccharomyces cerevisiae.

These findings support a dual role of the NTH of **Cdc25** in both enabling the **glucose** signal and being responsible for its attenuation.

The mammalian p140(ras-GRF) catalytic domain (CGRF) restores **glucose** signaling in S. cerevisiae only if tethered between the N-terminal half (NTH) of S. cerevisiae **Cdc25** and the C-terminal 37 amino acids.

We also show that 7 Ser to Ala mutations at the cAMP-dependent protein kinase putative phosphorylation sites within the NTH of **Cdc25** eliminate the descending portion of the **glucose** response curve, responsible for signal termination.

The regulatory domains in each Ras exchanger mediate the signals arriving from upstream elements such as tyrosine kinases for Sos, or Ca2+ and G proteins for p140.(Ras-GRF) In this study, we show that the N-terminal half (NTH) of S. cerevisiae **Cdc25**, as well as the C-terminal 37 amino acids, is essential for processing the elevation of cAMP in response to **glucose**.

The **Cdc25** protein of Saccharomyces cerevisiae is required for normal **glucose** transport.

In this paper it is reported that the **Cdc25** protein, in addition to its stimulatory role in the RAS/adenylate cyclase pathway, regulates **glucose** transport.

Cdc25 is not the signal receiver for **glucose** induced cAMP response in S. cerevisiae.

A crucial element of this model is that the exchanger, **Cdc25** is

activated by **glucose**.

We here show, in contrast to this view, that **Cdc25** cannot be the receiver of the **glucose** signal.

The **glucose** signal is processed by the Cdc25/Ras/adenylyl cyclase pathway, where the role of **Cdc25** is to catalyse the GDP-GTP exchange on Ras.

Phosphorylation of the S. cerevisiae **Cdc25** in response to **glucose** results in its dissociation from Ras [published erratum appears in Nature 1993 Jan 21;36(6409):278].

We report here the use of highly selective anti-Cdc25 antibodies to demonstrate that **Cdc25** is a phospho protein and that in response to **glucose** it is hyperphosphorylated, within seconds, by the cyclic AMP-dependent **protein kinase**.

This result demonstrates the requirement of **CDC25** for mediation of **glucose** signal transmission.

Our data suggest that the alpha domain of the **CDC25** protein is involved in **glucose** signal transduction, whereas the beta 2 domain is required for downregulating the cAMP control chain.

Functional mapping of the cell cycle START gene **CDC25** has revealed two domains which are dispensable for viability (germination and growth in **glucose** media), but are essential for sporulation and differentially involved in glucose-induced cAMP signaling.

The Saccharomyces cerevisiae start mutant carrying the **cdc25** mutation is defective in activation of plasma membrane ATPase by **glucose**.

To test whether Ras-15A and Ras-17N interfere with Ras function by blocking GDP-GTP exchange **proteins**, we examined their physical interaction with the **CDC25** exchange protein.

The **CDC25** gene from S. cerevisiae encodes an activator of Ras **proteins**.

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Here, we describe mutational analysis of Ha-ras for the identification of residues critical for the ability of Ras to interact with **Cdc25** and related guanine nucleotide-release **proteins**.

A growing number of genes from various organisms have been postulated to encode GDSs on the basis of sequence similarity with the Saccharomyces cerevisiae **CDC25** gene, whose product acts as a GDS of RAS **proteins**.

Isolation and nucleotide sequence of a Saccharomyces cerevisiae **protein kinase** gene suppressing the cell cycle start mutation **cdc25** .

Our data suggest that the **cdc25** suppressor gene encodes a cAMP-independent **protein kinase** involved in the control of the cell cycle start.

On the basis of the structure of cdk2/CksHs1 complex and on our kinetic results, we propose that the binding of Cks proteins to C-lobe of cdk2 is stabilized by the presence of **cyclin** A and that it may modify the orientation of the loop carrying residues 14 and 15 and their consequent access for dephosphorylation by **cdc25** phosphatases.

The cellular content of **Cdc25**p, the Ras exchange factor in Saccharomyces cerevisiae, is regulated by destabilization through a **cyclin** destruction box [published erratum appears in J Biol Chem 1995 Oct 27;270(43):26020].

The amino-terminal part of **Cdc25**p has a sequence similar to the **cyclin** destruction box (CDB) of mitotic **cyclins**.

The **CDC25** gene product is a guanine nucleotide exchange factor for **Ras proteins** in yeast.

The function of the mutant proteins was tested in vivo in both a Saccharomyces cerevisiae **cdc25** complementation assay and in a mammalian fos-luciferase assay, and in in vitro assays on human and yeast **Ras proteins**.

Influence of **guanine nucleotides** on complex formation between Ras and **CDC25** proteins.

Extracts of strains containing high levels of **Cdc25**p catalyze both removal of **GDP** from and the concurrent binding of **GTP** to Ras.

Increasing proportions of **GTP** bound to the various ras proteins correlated with increasing biological potency to bypass **cdc25** lethality in yeast.

Yeast **cdc25** phosphatase, which is specific for removal of phosphate from **tyrosine** at the active site of p34cdc2 enzyme, was expressed in bacteria and caused extensive in-vitro activation of p13suc1-purified enzyme from pith and suspension cells cultured without cytokinin.

Degradation of **Cdc25**p and CDB containing **beta-galactosidase** was found to be independent of various cell cycle arrest points.

Oligonucleotide primers derived from a mouse cDNA sequence homologous to the Saccharomyces cerevisiae **CDC25** gene product were used to screen a human **brain** cDNA library.

These data suggest that **Cdc25** might not be required in certain conditions for the guanine nucleotide exchange reaction in Ras and that it might be implicated in anchoring the Ras/adenylate cyclase system to the **plasma membrane**.

The results suggest that the **Cdc25** protein is tightly associated with the membrane but is not an intrinsic **membrane protein**, since only EDTA at pH 12 can solubilize the protein.

Using degenerate oligonucleotides that encode these conserved sequences, we have used **polymerase chain reactions** to amplify fragments of mouse and human cDNAs related to the yeast **CDC25 [?]** gene.

It is also demonstrated that, concomitantly with hyperphosphorylation, **Cdc25** partially relocalizes to the **cytoplasm**, reducing its accessibility to membrane-bound Ras.

The overexpression of the 3' terminal region of the **CDC25** gene of Saccharomyces cerevisiae causes growth inhibition and alteration of **purine nucleotides** pools.

top

Site-directed mutagenesis of the Saccharomyces cerevisiae **CDC25** gene: effects on mitotic growth and cAMP signalling.

The product of the START gene **CDC25**, an upstream element of the RAS/adenylyl cyclase pathway in Saccharomyces cerevisiae, was identified using specific **antibodies** raised against a chimeric beta-galactosidase/CDC25 protein.

Characterization, cloning and sequence analysis of the **CDC25** gene which controls the **cyclic AMP** level of Saccharomyces cerevisiae.

The **CDC25** "Start" gene of Saccharomyces cerevisiae: sequencing of the active C-terminal fragment and regional homologies with

rhodopsin and cytochrome P450.

A model of **Cdc25 [?]** phosphatase catalytic domain and Cdk-interaction surface based on the presence of a **rhodanese** homology domain.

Using the generalized profile technique, a sensitive method for sequence database searches, we found an extended and highly significant sequence similarity between the **Cdc25 [?]** catalytic domain and similarly sized regions in other proteins: the non-catalytic domain of two distinct families of MAP-kinase phosphates, the non-catalytic domain of several **ubiquitin** protein hydrolases, the N and C-terminal domain of **rhodanese**, and a large and heterogeneous groups of stress-response proteins from all phyla.

A 350-amino acid kinase domain at the C-terminal end shows high homology to the catalytic domains of **protein kinase** A, **protein kinase** C, S-6 kinase of Xenopus, and the suppressor of **cdc25** of yeast.

Although P. carinii **Cdc25 [?]** could also restore the **DNA damage** checkpoint in cdc25-22 cells, it was unable to restore fully the DNA replication checkpoint.

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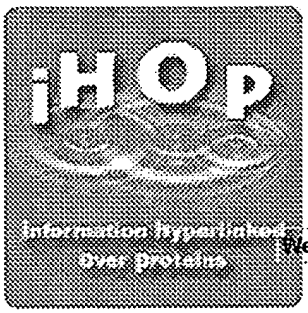

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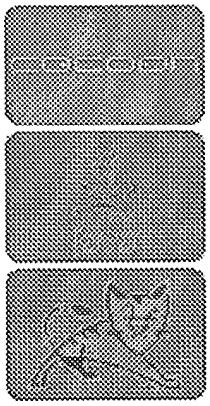

Symbol	Name	Synonym/ D
	[redacted]	Life cy
cdc25	**cdc25**	
CDC25		
CDC25C	cell division cycle 25C	
CDC25A	cell division cycle 25A	
CDC25B	cell division cycle 25B	
Cdc25a	cell division cycle 25 homolog A (S. cerevisiae)	
Cdc25b	cell division cycle 25 homolog B (S. cerevisiae)	
Cdc25c	cell division cycle 25 homolog C (S. cerevisiae)	
Cdc25l	**CDC25**-like protein	
CDC25 Vstring		
RASGRF1	Ras protein-specific guanine nucleotide-releasing factor 1	**CDC25**
RASGRP2	RAS guanyl releasing protein 2 (calcium and DAG-regulated)	**CDC25**L
MARK3	MAP/microtubule affinity-regulating kinase 3	**Cdc25**C-ass protein kinas
Rasgrf1	RAS protein-specific guanine nucleotide-releasing factor 1	**CDC25**
Rasgrp2	RAS, guanyl releasing protein 2	**CDC25**L
stg		**cdc25**
twe		**cdc25**
cdc-25.1		**cdc25**.1
cdc-25.2		**cdc25**.2
cdc-25.4		**cdc25**.4
TPK1		**CDC25** supp protein kinas

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